EPA's Response to 12/21/01 Comments Received From W.R. Grace On The Supplement to The Export/Screening Plant Administrative Record

On December 21, 2001 W.R. Grace Co., ("Grace") submitted comments to EPA on the supplement to the Export/Screening Plant administrative record (the "AR"), which contains the documents that formed the basis for the Action Memorandum Amendment, dated July 20, 2001. The Action Memorandum Amendment authorized further response actions at the Export and Screening Plants and at several other locations in Libby.

Grace has previously submitted comments (September 28, 2000) on the administrative record which supported the initial decision to take response actions at the Export and Screening Plants (as reflected in the original Action Memorandum, dated May 23, 2000). EPA provided detailed written responses to those comments on July 26, 2001. As will be further discussed below, many of Grace's comments are repetitive of those previously submitted; thus, many of EPA's July 26, 2001 responses remain relevant in this response.

Overview

Grace characterizes EPA's cleanup actions performed at Libby, Montana as arbitrary and capricious. While Grace cannot ignore the overwhelming rate of asbestos-related death and disease in Libby, it argues that such conditions are a result of past exposure. Where EPA propounds evidence of current exposure, Grace attacks the science, methods and findings of such efforts. Grace's attacks are without merit.

The Comprehensive Environmental Response, Compensation and Liability Act (CERCLA, 42 U.S.C. § 9601 et. seq.,) was codified to provide EPA and other relevant agencies authority to remedy situations in which hazardous substances were released, or for which there was a threat of release into the environment. The statute is preventive in nature, providing EPA the capability to prevent future releases and to eliminate the likelihood of future exposure where such releases have

already occurred. CERCLA does not require EPA to prove that people are sick, nor that releases are or have been occurring. Rather, in the case of time-critical removal actions, such as Libby, EPA must make the determination, based on the factors in the National Contingency Plan (NCP), that there is a threat to public health or welfare or the environment, whether from an actual or threatened release of a hazardous substance. (See 40 C.F.R. § 300.415(b)). Prior to commencing actions at Libby, EPA considered each of the factors set forth in 40 C.F.R. § 300.415(b) and determined that those enumerated in its Action Memoranda were applicable and that a threat to public health did exist. In addition, while not required by the NCP, EPA performed quantitative and qualitative risk analysis to further understand the nature of the threat. All of EPA's work, including its review of analyses by others, including Grace, supports the response action decisions EPA has made for the Libby Asbestos Site. While Grace challenges many aspects of EPA's risk work, technical methodology and public health conclusions, it assiduously avoids challenging the applicability of the 300.415(b) factors.

Despite the fact that production of asbestos-contaminated vermiculite ceased in 1990, exposure to non-naturally occurring amphibole asbestos was prevalent when the EPA response team arrived in Libby in November 1999 and continues at unremediated properties today. Fifty percent of properties that EPA has sampled in Libby have had at least trace levels of amphibole asbestos in some areas of their soils, five percent have levels higher than one percent. Eighteen percent of the properties sampled to date have had detectable levels of amphibole asbestos dust inside of buildings, while sixty percent have Zonolite Attic Insulation (Libby vermiculite insulation), which ranges from non-detect to five percent amphibole asbestos. EPA has more than 2000 properties to sample.

It cannot be reasonably disputed that, because of the spread of various Libby vermiculite materials throughout the area, Libby residents are coming into contact with the non-naturally occurring amphibole asbestos. EPA has observed such contacts, surveyed Libby residents who report a multitude of such contacts and has performed simulations involving every-day activities that demonstrate such contacts.

ATSDR's study of mortality results shows that there is an unequivocal association between historic exposure to Libby amphibole asbestos and death from asbestos-related disease. The study found that a Libby resident is forty to sixty times more likely to die from an asbestos-related disease than the average American and the finding of one in two thousand deaths from mesothelioma in this community far exceeds the approximately one in one million cases which occur in the general population each year. The ATSDR health screening performed in 2000 and 2001 clearly shows a dose-response relationship between exposure to Libby amphibole asbestos and lung abnormalities. Of the people who were least exposed, i.e., those who reported no known contact with Libby vermiculite, five percent had lung abnormalities. For those who reported one or more exposures to Libby vermiculite, ATSDR reported a higher rate of abnormalities. In fact, ATSDR found that the greater the number of exposure pathways reported, the greater the proportion of lung abnormalities observed. ATSDR found that a minimum of eighteen percent of the population screened had lung abnormalities. Of particular importance, eighty percent of those found to have lung abnormalities did not work for Grace and fifty-four percent were neither Grace workers nor their family members.

Contrary to Grace's characterization of the observed abnormalities as "beauty spots", these abnormalities are not benign. In fact, the majority of medical research on asbestos-related disease and the observed pathology and progression of asbestos-related lung disease in Libby indicate that once exposed, an individual with lung abnormalities has a high probability of manifesting disease and impairment. Whether the disease is asbestosis, lung cancer or mesothelioma, the result is respiratory debilitation and/or death. The Libby Center for Asbestos Related Disease is currently treating over 800 patients for varying degrees of asbestos-related lung impairment and over the past three years has identified as many as twenty cases of mesothelioma among current or former Libby residents.

As described previously to Grace and as discussed herein, the work performed independently by ATSDR, Amandus, McDonald, and Lockey all show that exposure to amphibole asbestos causes lung disease which can be debilitating or deadly. The fact that those currently ill were, by virtue of the latency period, exposed ten or twenty years ago does not change the ultimate conclusion: Libby amphibole asbestos causes lung disease and death.

In summary, EPA has clearly followed the NCP in its conduct of removal evaluations and response actions in Libby. EPA has documented the release of a hazardous substance (Libby amphibole asbestos), described its nature and extent through a removal site evaluation and appropriately used the criteria found in 300.415(b)(2) in deciding what actions were and are still necessary in Libby.

EPA's response is divided into categories covering the issues on which Grace has commented. Grace's topic headings are italicized and, where possible, the page number is referenced from Grace's comments. Where Grace has attached exhibits to its comments, EPA has reviewed the exhibits in the context of the comments and responded accordingly.

I. *Occupational Exposures (page 3)*

Grace is concerned that EPA's determination of an endangerment is based on studies which reflect conditions that no longer exist in Libby today. In particular, Grace believes that the occupational exposures evaluated in these studies are much higher than any exposure which would be experienced by a current Libby resident. Thus, Grace concludes that past exposures caused the death and disease experienced in Libby today and that there is no evidence to suggest that anyone could become ill under current conditions.

Despite the fact that Grace has already made this point in its comments on the administrative record for the Export and Screening plants, EPA makes the following response in addition to those already provided. Grace misconstrues EPA's use of the published literature. While the conditions to which the subjects of these studies were exposed may or may not have been the same as those experienced today, the studies definitively show that exposure to Libby asbestos causes lung disease. Grace's polemic on the studies of McDonald (1986) (see Attachment 65 & 66) and Amandus (1987 a,b,c) (see Administrative Record #'s 335073, 338134, 338247) at Libby and the Lockey studies (see Administrative Record Supplement # 484310 and Administrative Record # 338245) conducted at the Scott's plant in Marysville, Ohio (commencing on page 3 of the comments) is a good example of such confusion.

The studies of Amandus (1987 a,b,c) (see Administrative Record #'s 335073, 338134,

338247) and McDonald (1986) (see Attachment 65 & 66) at Libby proved that exposure to Libby Asbestos can cause lung disease and death. While those studies included exposure duration and intensities which may, in some cases, be much higher than that which would normally be encountered in Libby today, the studies defined no lower bound at which such exposure may cause harm. The very same materials to which workers were exposed in the Amandus and McDonald studies is present in many locations in Libby today.

The Marysville study (Lockey et al., Pulmonary Changes After Exposure to Vermiculite Contaminated with Fibrous Tremolite, Am. Rev. Resp. Disease, 129:952-958, 1984) (see Administrative Record Supplement # 484310) defined three exposure groups: 1) a low exposure group, representative of background exposures in a normal U.S. uncontaminated area; 2) a medium exposure group which had "low level fiber exposure"; and 3) a high level exposure group. The medium group consisted of employees who worked in the warehouse, in packaging and in central maintenance. This group had a 5 % prevalence of lung abnormalities (Table #4, Page 955), twice that of the low exposure group and half that of the high exposure group. Thus, the study confirmed a exposure-response relationship between asbestos exposure and lung abnormalities.

In the response to Grace's first set of comments on the administrative record, EPA made the following statements:

> *It is interesting to note that the levels of amphibole asbestos* ***in solid media*** *to which Marysville workers were exposed is the same as or lower than that to which those at the Screening and Export Plants were exposed.* (Emphasis added.)
>
> *Both the sweeping scenario and Phase II investigations reveal that Libby residents may currently be exposed to levels of airborne amphibole asbestos fibers which are higher than those classified as the medium exposure group in the Marysville study, which showed a positive association between exposure and asbestos-related lung abnormalities.*

(page 4)

Grace challenges these facts. Preliminarily, Grace counters that EPA's use of phase contrast microscopy (PCM) in Libby can't be compared to the use of scanning electron microscopy (SEM) with energy dispersive X-ray and transmission electron microscopy (TEM). This comment is interesting for several reasons. First, solid media is measured with polarized light microscopy (PLM), not with any of the techniques discussed by Grace. Grace has not refuted that the **solid media** to which Marysville workers were exposed is any different than that to which Libby residents are exposed. Individuals at the Export and Screening Plants were faced with asbestos in solid media, sometimes as high as 35%, much greater than that faced by the Marysville workers who, even according to Grace, should have never faced greater than 7% in solid media, because Grace has argued that its concentrate never contained more than 7% asbestos, and usually less than that. Grace asserts that Lockey did not report solid media concentrations of asbestos, but Lockey does report it as 2% in his article entitled "Fiber Contamination of Vermiculites: A Potential Occupational and Environmental Health Hazard", Environmental Research 41, 207-218 (1986) (see Administrative Record #338254). Thus, individuals at the Export Plant were exposed to asbestos in solid media eighteen times that found at Marysville.

(page 5)

Second, Grace argues that Libby residents are not exposed to levels of amphibole asbestos as high as those experienced by Marysville workers and that the cumulative exposure of the workers is greater than that presented to residents. This is incorrect. EPA has measured higher concentrations of asbestos in both solid media and in air than found at Marysville. Solid media exposures were discussed above. Grace does not accurately portray the actual levels of airborne asbestos either at Libby or at Marysville. For example, levels of airborne asbestos in Libby were found to be higher than 4 f/cc (PCME), during the sweeping/bagging scenario, which reflects an activity that occurred on a daily basis at the Screening plant. If one assumes that sweeping only occurred for thirty minutes a workday and that a resident experienced no other asbestos exposures during the day, there would be a time-weighted average exposure of 0.25 f/cc. At Marysville, the range of time-weighted average exposures to the medium group was 0.031 f/cc to

.415 f/cc. Thus, Libby residents would be expected, in some cases, to have higher exposures than the workers in the medium exposure group at Marysville. It is worth noting that the Screening Plant exposure of 4.0 f/cc is four times higher than the short-term OSHA exposure limit and the 0.25 time-weighted average exceeds the OSHA permissable exposure limit.

It should be noted that Grace has asserted that its asbestos emissions were always below or near the occupational exposure limits at its facilities. Nevertheless, Grace's own records reflect that asbestosis affected 41% of Grace's Libby workers and 28% of workers at other facilities which processed Libby vermiculite (Woods) (see Administrative Record Supplement # 485797). The OSHA standard is recognized to allow for a calculated cancer risk of 3E-03, which is thirty times the exposure that EPA would allow for cancer risk at a Superfund site, even with risk management. EPA expects that exposures experienced by Libby residents would be longer than the eight-hour period normally used to set the OSHA occupational limits, thus rendering the OSHA limit even less protective.

Grace's use of the cumulative asbestos exposure (12.07 f/ml-yr) for employees with lung abnormalities confounds the issue. The Lockey study reveals that the range of exposure for those with lung abnormalities was as low as .01 f/ml-yr and as high as 39.9 f/ml-yr. It is important to note that the 39.9 f/ml-yr figure skews the mean towards higher levels. Assuming that the Parkers were to continue sweeping areas of the Screening Plant daily for thirty minutes for the seven years they owned the property prior to EPA's cleanup and that they experienced no other asbestos exposures during the remaining 23.5 hours of the day, their cumulative exposure would have been 1.75 f/ml-yr, much higher than some of those with lung abnormalities at Marysville. In addition, this exposure is higher than the mean exposure experienced by the medium exposure group at Marysville, which showed almost twice the abnormalities experienced by the control group. In reality, the Parkers, their children and grandchildren were exposed in many more ways than just sweeping. Other potential pathways included house cleaning, playing in soils, vehicle traffic, etc. If EPA had not intervened, the Parker family's cumulative exposure may have exceeded that of the high exposure group at Marysville. Indeed, ATSDR's screening of 6200 Libby residents found that 40% of those individuals were exposed to six or more asbestos exposure pathways. It is also particularly illuminating that Lockey indicates that the cumulative

fiber exposure at Marysville was low compared with that in other studies, below 10 f/ml-yr for 90% of the employees. Additionally, ninety percent of the employees had less than twenty years employment, indicating a very short latency period, i.e., a very short time period in which to observe the development of lung abnormalities in this population.

Grace's reliance on EPA's active cleaning scenario to suggest the risk in Libby is minimal is misplaced. Grace's use of the active cleaning scenario belies the fact that all of the cleanups performed under this administrative record concerned locations which had much higher levels of asbestos measured in both solid media and air than found in the active cleaning scenario. As discussed above, Lockey's work reflected an endangerment at asbestos levels such as those present at the removal locations. Residents do not simply have the one exposure from cleaning their home, but also live in their home and may have contacts around Libby with other vermiculite sources.

Finally, Grace argues that EPA exaggerates the clinical findings at Marysville. Lockey found 4.4% of the those with X-rays showed clinical findings of lung abnormalities consistent with asbestos exposure. These findings were in a young population (mean age less than 42) with a relatively short exposure period and very short period in which to observe clinical effects. EPA's analysis is anything but an exaggeration. Southern Lincoln County has approximately 10,000 residents. Following Grace's logic, it would be acceptable to have 440 such residents diseased from asbestos exposure. One should note that ATSDR has already identified four times this percentage, totaling approximately 1200 individuals, showing lung abnormalities in this area. This is not surprising, as the ATSDR study provided the opportunity to observe the expression of clinical findings over a longer period of time.

It is also interesting to note the use of SEM by Lockey, which Grace characterizes as an unapproved and little used methodology for identifying asbestos fibers. As previously indicated, many scientists, including Grace scientists, use SEM in asbestos analysis.

II. *Mortality Study (page 5)*

The ATSDR mortality study shows the relationship between asbestos-related death and historical asbestos exposure in the community. While Grace quotes ATSDR's purpose from a paragraph later in the mortality study, it conveniently skips the language in the first paragraph which indicates the relationship between historic exposures and current medical conditions. In a recent communication with Mr. Steve Dearwent (author of the ATSDR mortality study), he stated that a recent update and re-analysis of the Libby mortality data found that asbestosis rates were substantially higher than previously reported and that lung cancer rates were now determined to be statistically significantly elevated over expected rates.

Grace emphasizes what it perceives as a lack of separation between occupational and non-occupational exposures in the mortality study. This point is irrelevant, as it is beyond the scope of what ATSDR intended to do and the purposes for which the study has been used by EPA. The ATSDR mortality study clearly shows three points: first, there is an unequivocal association between historic exposure to Libby amphibole asbestos and death from asbestos-related disease; second, the rate of asbestos-related deaths in Libby far exceeds asbestos-related deaths in almost any other community; and third, the finding of one in two thousand deaths from mesothelioma in this study exceeds the one in one million rate expected in the general population by three orders of magnitude. The mortality study illustrates that the hazardous substance in question causes disease and death. While occupational exposures may not continue at former Grace facilities in Libby, similar levels of exposure may be occurring routinely in this community. The ATSDR mortality study is further bolstered by a recent NIOSH evaluation of counties in the country with the highest mortality rates of asbestosis. The rates of asbestosis identified by NIOSH in Lincoln County were similar to those found in the ATSDR study. Libby was found by NIOSH to be among those communities having the highest rates of asbestosis in the nation.

III. *ATSDR Medical Screening Report of August 23, 2001 (page 8)*

- *A. Study Design*
- *B. Results*
- *C. Discussion of Findings*

1. ATSDR's Screening Results are not Diagnostic of Any Asbestos-Related Disease

The ATSDR study shows a very high prevalence (conservatively estimated at 18%) of asbestos-related lung abnormalities among the participants in the study, which constituted approximately 60% of the southern Lincoln County population. While ATSDR's regression analysis can only statistically link six exposure pathways to clinical findings, the report also indicates that other exposure pathways are contributing to disease. ATSDR specifically finds that 90% of the people in the surveyed group had two or more such exposures, and that all of the exposures pathways had odds ratios suggesting increased risk. In addition, those not identifying any exposure pathways during the ATSDR interview showed 5% lung abnormalities. Thus, Grace's attempt to analyze each exposure pathway separately is misleading. Occupational exposure is not the sole cause of asbestos-related disease in Libby. In fact, 69% of those having lung abnormalities had no occupational exposure at all. Further, a recently completed case-series by ATSDR and US Public Health Service, confirmed eight cases of non-occupational asbestos-related lung abnormalities among a subgroup of twenty-two Libby patients being followed by a Spokane pulmonologist (Dr. Whitehouse). This case-series found no past medical, fibrogenic dust exposure or occupational history to account for these individuals lung abnormalities. Confirmation of asbestos-related lung disease from purely environmental pathways is exceedingly rare, especially in the U.S., and underscores the particularly hazardous situation present in Libby.

ATSDR's study clearly shows the following four important points: 1) there are ongoing frequent exposures to amphibole asbestos reported by the study participants; 2) 90% of the study participants have multiple exposure pathways, 40% have six or more such pathways; 3) there are several non-occupational exposures that are clearly associated with clinical findings; and 4) behaviors resulting in contact with amphibole asbestos result in clinical findings.

2. *The results do not indicate that current conditions are causing pleural findings.*

(page 11)

Grace argues that ATSDR's exposure pathways no longer exist. This is simply not true. Up until EPA's cleanup actions, Libby residents were contacting amphibole asbestos at the Export plant or Screening plant, adults and children were contacting vermiculite piles and tailings at Plummer Elementary School and there was direct contact with vermiculite ores or waste (and its associated amphibole asbestos) at all the locations where EPA has taken such actions, including the high school and middle school. As to subsurface contamination, the Agency has chosen to take the reasonable step of removing source materials such as those underneath the high school track where they may cause harm in the immediate future. While Grace denies any risk from subsurface contamination, the Agency has been informed that contact with this material is happening now and is likely to occur in the future. For example, maintenance workers at the high school routinely encounter the material while maintaining the sprinkling system and would most certainly encounter it while repairing the track itself. At the Screening Plant, the Parkers regularly planted trees or performed infrastructure maintenance that brought them into contact with subsurface materials, including contaminated vermiculite.

3. *Limitations of Study.*

a. *Use of Volunteer Study*

(page 12)

Grace implies that voluntary participation in the ATSDR study biases the findings. Grace fails to convey that: 1) 61% of the population is a very significant participation rate; 2) approximately 1,700 more people have undergone testing, thus raising the percentage even more; 3) 994 people with lung abnormalities in such a small population is extremely significant; and 4) even if the remaining population participated and had no abnormalities, abnormalities would still appear in 11% of the population, a finding of great public health consequence. It is worth noting that ATSDR has combined medical screening results from the summer of 2000 and the summer of 2001 to arrive at a new combined data set. The new population of those tested is 7304, which is

72% of the Libby division of Lincoln County. Many of those not tested were children or did not meet the testing qualification criteria (i.e., did not live or work in Libby prior to 1990). Information from this additional screening is currently being evaluated.

Grace also spends much time mischaracterizing statements of individuals and organizations concerning the meaning of the ATSDR studies, for instance the letter of Pat Cohan. Statements to individuals indicating that they should seek medical advice before being fearful of medical disease have nothing to do with the meaning of the ATSDR study. Statements of the Agency concerning the lack of asbestos in Libby ambient air are not indicative of other exposure pathways currently present in Libby.

b. Lack of Control Group

(page 13)

Grace's concerns about the lack of a control group are misplaced. As ATSDR indicated in its published report:

> *The program was not designed as an analytic epidemiologic study with comparison groups and random sampling of exposed and comparison groups. Nevertheless, the data collected provide important information about the prevalence and degree of asbestos-related abnormalities among a large number of current and former Libby residents, and about the possible relationships between these abnormalities and a number of exposure pathways reported by community members.*

c. Reader Variability and Bias

(page 13)

Grace implies that the B-readers were biased. There is no reason to believe this to be true. The three B-readers are respected experts in their field and distinguished members of academic institutions. The readers were blinded to the identities of individuals who were screened. They have no incentive to misread the x-rays. In commenting on "reader variability and bias", Grace has failed to acknowledge the information in ATSDR's report about limitations on observer bias

(see p.26). Even though no external control group was available, internal comparisons within the study population are entirely consistent with expected exposure-response relationships. For example, only 5% of those with no apparent exposures had pleural abnormalities as compared to 11% for those with one to three exposure pathways, 15% for those with four to five exposure pathways, and 24% for those with six or more pathways. Furthermore, with respect to individual exposure pathways which individuals graded between never, sometimes, and frequently, the odds ratios increased with increasing frequency of the activity for five out of six pathways evaluated (handled vermiculite insulation, recreational activities along Rainy Creek, played at ballfields near expansion plant, played in vermiculite piles, and popped vermiculite). In addition, comparison of the prevalence of pleural abnormalities between Libby and other North American populations shows Libby to be markedly increased. (Studies of differing groups within the United States believed to have no substantive work-related asbestos exposures have found the prevalence of pleural abnormalities ranging from 0.2% among blue-collar workers in North Carolina (Castellan 1985) (Attachment 1), to 0.9% among loggers in Washington and Oregon (Stilbolt 1991) (Attachment 2) , to 1.8% among New Jersey residents (Anderson 1979) (Attachment 3 and (Administrative Record # 344137), and 2.3% among patients at Veterans Administration hospitals in New Jersey (Miller JA 1996) (Attachment 4)..

d. Significance of Obesity and Use of Oblique Films

(page 14)

Grace also implies that the study is invalid because of the extra views used to observe lung abnormalities and because of the prevalence of obesity in the community. These implications are unfounded. Even if ATSDR had limited its views to those traditionally used in epidemiological studies of asbestos exposure (posterior and anterior chest view only), there would still be a finding of 14% lung abnormalities. In addition, oblique views are widely used for observation of asbestos-related abnormalities in clinical practice for evaluation and were recommended for this study by a panel of medical experts. Beyond the ATSDR study, such recommendations persist. For example, a recent medical study recommends the use of obliques for surveillance studies

where both parenchymal and pleural changes are anticipated. ("Reliability and Validity of Chest Radiograph Surveillance Programs", Chest, Volume 120, pages 64-68, Attachment 5).

While Body Mass Index (BMI) may be a potential confounder for evaluations of pleural disease, very few epidemiologic studies have even considered the effect of BMI on their findings. The steps taken by ATSDR to include and account for BMI in their study design show that every effort was made to be more thorough, thoughtful, and conservative in their approach towards medical screening of the Libby population. The fact that BMI was not considered in the evaluation of other comparative populations suggests that actual percentage of true asbestos-related pleural abnormalities would be proportionally less in these populations, as well. Further, the ATSDR multivariate analysis was specifically adjusted for BMI and still found numerous statistically significant associations between vermiculite exposure pathways and asbestos-related pleural disease. Evaluation of the data set from 2000 medical screening period reveals that even if all participants with a BMI over 30 that were found to have asbestos related pleural abnormalities were entirely excluded from consideration, there would still be over 500 participants remaining with asbestos-related pleural abnormalities identified by at least 2 B-readers. Grace contends that the B-readers were biased and were over-reading x-rays secondary to BMI or fat. All B-readers were blinded to the identity of x-rays they were reading. Thus, if x-rays readings were biased or systematically being overread, then it is unlikely that consistent internal exposure-response relationships would be observed. Again the internal comparisons found by ATSDR within the study population are entirely consistent with expected exposure-response relationships. For example, only 5% of those with no apparent exposures had pleural abnormalities as compared to 11% for those with one to three exposure pathways, 15% for those with four to five exposure pathways, and 24% for those with six or more pathways. Furthermore, with respect to individual exposure pathways which individuals graded between never, sometimes, and frequently, the odds ratios increased with increasing frequency of the activity for five out of six pathways evaluated (handled vermiculite insulation, recreational activities along Rainy Creek, played at ballfields near expansion plant, played in vermiculite piles, and popped vermiculite).

Furthermore, ATSDR is currently completing a secondary evaluation among 300 participants with vermiculite exposure, symptoms, and normal or questionable radiographic findings on chest x-ray. This followup study uses high resolution CT scans to determine the percentage of individuals with asbestos related abnormalities that were missed by the chest x-rays. It is well established that chest x-rays are fairly insensitive in picking up many asbestos-related abnormalities, entirely missing all abnormalities in large percentages of individuals found to have asbestos-related abnormalities on CT scans, especially High Resolution CT scans, and histopathological evaluation. In one study, chest x-rays entirely missed interstitial abnormalities in 18% of those with histological evidence of asbestosis. The authors concluded that negative chest radiographs do not exclude interstitial fibrosis in a substantial percentage of workers with previous asbestos exposure (Kipen,HM, Lilis R, Suzuki Y, et. al. Pulmonary fibrosis in asbestos insulation workers with lung cancer: a radiological and histopathological evaluation. Br J Ind Med. 1987;44:96-100) (Attachment 6 and Administrative Record # 487149). In another study of shipyard workers chest x-rays missed pleural plaques in 49% and interstitial fibrosis in 27% of those evaluated, compared to high resolution CT scan (Neri S et al Asbestos-related lesions detected by high-resolution CT scanning in asymptomatic workers. Specificity, relation to the duration of exposure and cigarette smoking. Clin Ter. 1994;145:97-106). (Attachment 7) Thus, even if it is assumed that there is reader variability and over-reading due to the confounding presence of fat on some of the x-rays, as Grace contends, it is quite likely that the findings of 18% with asbestos-related pleural abnormalities is actually a conservative estimate of those with actual asbestos-related abnormalities, due to the insensitivity of x-rays.

e. No Discussion of Severity of Findings

(page 16)

Grace believes that because the ATSDR study does not differentiate the types of x-ray abnormalities, it cannot be compared with other studies that do. While the ATSDR study did not differentiate the types of pleural radiographic abnormalities found, it is clear that both asbestos-related (as determined by at least two of three expert B-readers) pleural and interstitial abnormalities were found in this population, with pleural abnormalities found in approximately

18% of those evaluated. Review of ATSDR data (unpublished but provided to Grace in discovery) and reports by treating physicians reveal that pleural abnormalities range from small circumscribed pleural plaques to diffuse bilateral pleural disease. Treating physicians have also reported a number of cases that have only pleural abnormalities on chest x-rays but were found to have substantial functional impairment, which was elucidated with more sophisticated medical testing than the simple spirometry which was used during the screening process. Further, Grace asserts that no exposure-response relationship can be observed in the ATSDR study. As previously discussed, this is not the case. The prevalence of asbestos-related abnormalities clearly follows exposure-response patterns throughout the ATSDR study and is entirely consistent with EPA's observations in Libby and elsewhere, which show that the greater the frequency and duration of contact with amphibole contaminated vermiculite, the greater the likelihood of associated disease.

4. Dr Weis' Discussion of Study

(page 16)

Grace argues that all of the studies cited by Dr. Weis in his discussion of ATSDR's report concerned "workers with significantly higher exposures to asbestos." While EPA does not have information about the actual exposure levels of individuals tested by ATSDR, EPA does know 1) these individuals had, and continue to have, multiple pathways of exposure, 2) that they have high levels of radiographic abnormalities stemming from those exposures, 3) that together their health statistics constitute one of the highest asbestosis and mesothelioma rates in the nation stemming from exposure, and 4) according to treating physicians, they have substantial ongoing morbidity stemming from their exposures. Given these facts, these individuals may have exposures similar to workers in the studies (see earlier discussion of the Lockey study). In fact, Erlich (1992) (Attachment 8) found that an average of 37% of workers with less than 1 month of exposure to amosite asbestos had pleural disease. He also found that no cumulative exposure threshold (ie. a safe level of exposure) for pleural and interstitial disease could be detected and that progression of pleural and interstitial disease were still detectable greater than 20 years after the end of exposure. We agree with Grace's comment from Hillerdal (1997) (see Administrative Record Supplement #

484253), that cumulative asbestos dust exposure is significantly associated with progression of pleural disease. This finding underscores the need for EPA actions to reduce any substantial asbestos exposures remaining in the Libby community, thus reducing their cumulative exposures. Also, with regard to Hillerdal (1997) (see Administrative Record Supplement #484253), Grace appears to disregard the remainder of Dr. Hillerdal's comments which state that:

> "Ten percent of the persons with non-malignant asbestos-related pleural lesions without signs of parenchymal fibrosis will develop radiological and clinical evidence of it in a 10-year period. Slightly restrictive lung function has been reported for groups with asbestos-induced pleural lesions; the principal determinant of this restrictive lung function is probably parenchymal inflammation or fibrosis. In careful pathologic investigations, small lesions in the bronchioles and surrounding parenchyma can be found in most patients with pleural plaques".

Grace implies that because the articles cited are not strictly related to tremolite, they have less value in understanding the progressive nature of the lung abnormalities at Libby. While the cited studies may not specifically focus on tremolite asbestos exposures, these studies show that abnormalities associated with various types of asbestos exposures can be progressive and can lead to more severe disease and impairment. Exposure to amphibole asbestiform minerals in this population is clearly consistent with pathologic disease seen associated with other forms of asbestos exposure. The exposures seen in Libby have been demonstrated to result in excess asbestos-related death and disease (occupational studies by NIOSH and McDonald, mortality studies, reports by treating physicians) as seen with other forms of asbestos exposure. In a review of several studies, NIOSH (Stayner L, Dankovic DA, Lemon R. 1996. "Occupational exposure to chrysotile asbestos and cancer risk: A review of the amphibole hypothesis" Am. J. Pub. Hlth. 86:107-114) (Attachment 9 and Administrative Record #487096) found that the tremolite exposures associated with mining in Libby may be among the most potent exposures related to asbestos risks for mesothelioma.

(page 17)

Grace contends that the articles cited by Dr. Weis do not support the "theory" of progression. All the articles cited by Dr. Weis reported that various asbestos-related abnormalities, whether pleural or interstitial, continued to progress over time regardless of the type of asbestos workers were exposed to. Again the study by Erlich (1992) (Attachment 8), which found that an average of 37% of workers with less than 1 month of exposure to amosite (another amphibole) had progression of pleural and interstitial disease which was still detectable greater than 20 years after the end of their exposure, clearly illustrates this point. Erlich also found that no cumulative exposure threshold (i.e. a safe level of exposure) for pleural and interstitial disease could be detected. Specifically, with respect to exposure to Libby asbestiform minerals, the recently reported case by Wright, et al (Wright RS, Abraham JL, Harber P, Burnett BR, Morris P, West P. Fatal Asbestosis 50 Years after brief high intensity exposure in a vermiculite expansion plant. Am J Respir Crit Care Med. 2002;165(8):1145-9) (Attachment 10) clearly documents progressive and fatal asbestos disease in a man 50 years after only a 2 month exposure to Libby vermiculite when he was 17 years old.

Further, a study being prepared for publication by Dr. Alan Whitehouse (Attachment 11) found statistically significant progressive loss of lung function among 67 patients from Libby with only asbestos-related pleural abnormalities identified on either chest x-ray or CT scan.
A recent case series completed by ATSDR and the US Public Health Service of twenty-two patients being followed by Dr. Whitehouse confirmed that asbestos-related radiographic lung abnormalities were occuring in individuals without any history of occupational asbestos exposures, household contact with former workers, or other confounders (i.e., past medical problems) which could cause mischaracterization of radiographic findings. (Attachment 12)

The pleural manifestations associated with exposure to various forms of asbestos range from effusions, circumscribed disease, and diffuse disease with variance depending on latency, idiosyncratic reactions and nature of exposure (dose & duration). "Pleural plaques are the most common manifestation of asbestos exposure and only rarely do they occur in persons who have no history or evidence of asbestos exposure" (Cotran RS, Kumar V, Collins T. Robbins pathologic basis of disease, 6th edn. Philadelphia: WB Saunders Company, 1999:, 732-4.)

(Attachment 13) The pleural abnormalities identified among those that participated in the medical screening program in Libby range from discrete or circumscribed pleural plaques to diffuse pleural disease. While these abnormalities may have been historically viewed as non-symptomatic markers of asbestos exposure, there is now a large body of scientific literature collected over the last 20 years that demonstrates that asbestos-related radiographic findings of circumscribed and diffuse pleural thickening represent observable evidence of disease processes associated with functional lung impairment and clinical symptoms. Furthermore, the presence of these radiographic findings appears to be associated with increased risk of mesothelioma and perhaps lung cancer.

While a few studies have not detected any associations between circumscribed pleural disease and functional impairment, by and large, the majority of peer reviewed epidemiologic studies, especially those performed more recently, have shown that circumscribed pleural disease is associated with increased symptoms and functional impairment. Also, the findings of a number of these studies should be given greater consideration as they involved populations large enough to detect statistical associations, had external controls, and accounted for potential confounders such as age, latency, cigarette smoking, and interstitial profusion. Grace's assertion that the medical consensus (citing Hillerdal 1978 (Attachment 14), Jones 1988 (Attachment 15), Murphy 1987 (Attachment 16), Gaensler (Attachment 17 and Grace exhibit 23), and Churg (Administrative Record # 487085) that pleural plaques are an isolated pleural finding that are not associated with significant reductions in pulmonary function (i.e., beauty spots) is plainly false. Grace misrepresents Hillerdal (1978) (Attachment 14), which was a radiographic study only and did not evaluate the relationship between pleural disease and functional impairment. However, Dr. Hillerdal did make the following comments based on his findings: "The formation of pleural plaques must be considered as a dynamic process, following inhalation of asbestos fibers, which initiates a slowly fibrosing, localized lesion of the parietal pleura. Once started, the lesion tends to progress and may finally calcify extensively. The progression is not affected by stopping the exposure". In a later study in which Dr. Hillerdal (1990) (Attachment 18) did evaluate lung function and radiographic abnormalities, he found that individuals with plaques had slightly lowered lung function compared to reference subjects. He also found that diffuse bilateral pleural

disease was associated with a more marked decrease in pulmonary function. Jones (1988) (Attachment 15), cited by Grace, is purely a review of various studies prior to 1988. Numerous studies, many of which address the authors' criticisms of the older research, have been completed subsequently and consistently have found impaired lung function associated with both circumscribed and diffuse pleural disease. Grace also submits a non-peer reviewed, non-published paper by a Dr. Gaensler (Attachment 17 and Grace exhibit 23) of Boston University as evidence that asbestos-related pleural disease is "harmless scurrilous beauty marks on the chest film". Grace cites that this paper was used in a litigation proceeding (exhibit 22) as apparent evidence of its validity. This paper provides no knowledge or specific information concerning the Libby situation or findings. The author's potential biases and purpose for writing this paper, which reviews the medical literature prior to 1991, are unknown. Grace also submits a short letter published in the journal American Review of Respiratory Disease in 1987 by Dr. Raymond Murphy (exhibit 21) to support the assertion that plaques are nothing more than spots on a lung x-ray and do not cause loss of function or symptoms. In his letter Dr. Murphy clearly recognized the importance of pleural abnormalities but felt that pleural plaques did not result in loss of function or symptoms citing a 1972 German reference. Also, Dr. Murphy recognized that diffuse pleural thickening may impair lung function and may lead to an "imprisoned lung' requiring decortication. Again, the consistency of findings in numerous studies over the last 20 years contradict assertions that plaques do not cause loss of function or symptoms. Some of the more notable studies that have found associations between pleural plaques and functional impairment and symptoms of breathlessness include the following:

* Bourbeau J, Ernst P, Chrome J, Armstrong B, Becklake MR The relationship between respiratory impairment and asbestos-related pleural abnormality in an active work force. Am Rev Respir Dis. 1990 Oct;142(4):837-42. (Attachment 19)
* Hilt B, Lien JT, Lund-Larson PG. Lung function and respiratory symptoms in subjects with asbestos-related disorders: a cross sectional study. Am J Ind. Med 1987; 11:517-528. (Attachment 20)
* Hillerdal G, Malmberg P, Hemmingsson A. Asbestos-related lesions of the pleura: parietal plaques compared to diffuse thickening studied with chest roentgenography, computed tomography, lung function, and gas exchange. Am J Ind Med. 1990;18(6):627-39. (Attachment 18)
* Schwartz DA, Fuortes LJ, Galvin JR, Burmeister LF, Schmidt LE, Leistikow BN, LaMarte FP, Merchant JA. Asbestos-induced pleural fibrosis and impaired lung function. Am Rev Respir Dis. 1990 Feb;141(2):321-6. (Attachment 21)
* Kouris SP, Parker DL, Bender AP, Williams AN. Effects of asbestos-related pleural disease on pulmonary

function. Scand J Work Environ Health. 1991 Jun;17(3):179-83. (Attachment 22)

* Britton MG. Asbestos pleural disease. Br J Dis Chest. 1982;76:1-10. (Attachment 23)
* Kilburn KH, Warshaw R. Pulmonary functional impairment associated with pleural asbestos disease. Circumscribed and diffuse thickening. Chest. 1990 Oct;98(4):965-72. (Attachment 24)
* Jarvholm B, Sanden A. Pleural plaques and respiratory function. Am J Ind Med. 1986;10(4):419-26. (Attachment 25)
* Lilis R, Miller A, Godbold J, Chan E, Benkert S, Selikoff IJ. The effect of asbestos-induced pleural fibrosis on pulmonary function: quantitative evaluation. Ann N Y Acad Sci. 1991 Dec 31;643:162-8. (Attachment 26)
* Schwartz DA. The clinical relevance of asbestos induced pleural fibrosis. Ann N Y Acad Sci. 1991 Dec 31;643:169-77. (Attachment 27)
* Ernst P, Bourbeau J, Becklake MR.Pleural abnormality as a cause of impairment and disability. Ann N Y Acad Sci. 1991 Dec 31;643:157-61. (Attachment 28)
* Hedenstierna G, Alexandersson R, Kolmodin-Hedman B, Szamosi A, Tollqvist J.Pleural plaques and lung function in construction workers exposed to asbestos. Eur J Respir Dis. 1981;62(2):111-22. (Attachment 29)
* Oliver LC, Eisen EA, Greene R, Sprince NL. Asbestos-related pleural plaques and lung function. Am J Ind Med. 1988;14(6):649-56. (Attachment 30)
* Miller A, Lilis R, Godbold J, Chan E, Selikoff IJ. Relationship of pulmonary function to radiographic interstitial fibrosis in 2611 long-term asbestos insulators. Am Rev Resp Dis 1992; 145:263-270. (Attachment 31)
* Hillerdal G, Malmberg P, Hemmingsson A. Asbestos-related lesions of the pleura: parietal plaques compared to diffuse thickening studied with chest roentgenography, computed tomography, lung function, and gas exchange. Am J Ind Med. 1990;18(6):627-39. (Attachment 18)

In general, diffuse pleural disease has been associated with more severe functional impairment and symptoms than circumscribed pleural disease. Studies that have found associations between diffuse pleural disease and functional impairment and symptoms of breathlessness or dyspnea, where evaluated, include the following:

* Bourbeau J, Ernst P, Chrome J, Armstrong B, Becklake MR The relationship between respiratory impairment and asbestos-related pleural abnormality in an active work force. Am Rev Respir Dis. 1990 Oct;142(4):837-42. (Attachment 19)
* Hillerdal G, Malmberg P, Hemmingsson A. Asbestos-related lesions of the pleura: parietal plaques compared to diffuse thickening studied with chest roentgenography, computed tomography, lung function, and gas exchange. Am J Ind Med. 1990;18(6):627-39. (Attachment 18)
* Schwartz DA, Fuortes LJ, Galvin JR, Burmeister LF, Schmidt LE, Leistikow BN, LaMarte FP, Merchant JA. Asbestos-induced pleural fibrosis and impaired lung function. Am Rev Respir Dis. 1990 Feb;141(2):321-6. (Attachment 21)
* Kouris SP, Parker DL, Bender AP, Williams AN. Effects of asbestos-related pleural disease on pulmonary function. Scand J Work Environ Health. 1991 Jun;17(3):179-83. (Attachment 22)
* Britton MG. Asbestos pleural disease. Br J Dis Chest. 1982;76:1-10. (Attachment 23)
* Kilburn KH, Warshaw R. Pulmonary functional impairment associated with pleural asbestos disease. Circumscribed and diffuse thickening. Chest. 1990 Oct;98(4):965-72. (Attachment 24)
* Schwartz DA. The clinical relevance of asbestos induced pleural fibrosis. Ann N Y Acad Sci. 1991 Dec 31;643:169-77. (Attachment 27)

* Ernst P, Bourbeau J, Becklake MR.Pleural abnormality as a cause of impairment and disability. Ann N Y Acad Sci. 1991 Dec 31;643:157-61. (Attachment 28)
* Miller A, Lilis R, Godbold J, Chan E, Selikoff IJ. Relationship of pulmonary function to radiographic interstitial fibrosis in 2611 long-term asbestos insulators. Am Rev Resp Dis 1992; 145:263-270. (Attachment 31)
* Hillerdal G, Malmberg P, Hemmingsson A. Asbestos-related lesions of the pleura: parietal plaques compared to diffuse thickening studied with chest roentgenography, computed tomography, lung function, and gas exchange. Am J Ind Med. 1990;18(6):627-39. (Attachment 18)
* Rosenstock L, Barnhart S, Heyer NJ, Pierson DJ, Hudson LD. The relation among pulmonary function, chest roentgenographic abnormalities, and smoking status in an asbestos-exposed cohort. Am Rev Respir Dis. 1988 Aug;138(2):272-7. (Attachment 32)
* Rosenstock L. Roentgenographic manifestations and pulmonary function effects of asbestos-induced pleural thickening. Toxicol Ind Health. 1991 Jan-Mar;7(1-2):81-7. (Attachment 33)
* Kee SL, Blanc P. Causes of pulmonary impairment in asbestos-exposed individuals with diffuse pleural thickening. Am J Resp Crit Care Med. 1996;154:789-93. (Attachment 34)
* Lilis R, Miller A, Godbold J, Selikoff IJ. Radiographic abnormalities in asbestos insulators: effects of duration from onset of exposure and smoking. Relationship of dyspnea with parenchymal and pleural fibrosis. Am J Ind Med. 1991; 20:1-15. (Attachment 35)
* Miller A, Teirstein AS, Selikoff IJ. Ventilatory failure due to asbestos pleurisy. Am J Med. 1983 Dec;75(6):911-9. (Attachment 36)
* McGavin CR, Sheers G. Diffuse pleural thickening in asbestos workers: disability and lung function abnormalities. Thorax. 1984 Aug;39(8):604-7. (Attachment 37)

The studies cited above have typically involved individuals with occupationally-related asbestos exposures. Grace argues that since these exposures were occupationally related they were higher than exposures experienced by those in Libby and thus not applicable or comparable. This assertion is unfounded as we know very little about the cumulative non-occupational exposures experienced among those living in Libby. However, we do know that residents have had 1) multiple pathways of exposure, 2) that direct contact with vermiculite can generate exposures easily exceeding occupational standards, and 3) these cumulative exposures have led to high rates of asbestos-related pulmonary disease and mortality among individuals in the Libby community. Additionally, as opposed to worker cohorts, exposures in Libby may start in infancy potentially resulting in lifetime cumulative exposures equivalent to or even surpassing occupational exposures. This is especially important since the earlier in life you are exposed to asbestos, regardless of dose, the more likely you are to experience asbestos-related morbidity and mortality.

It is well established that asbestos exposure results in non-malignant fibrotic lung disease and malignancies of lung cancer and mesothelioma. The ATSDR mortality research confirms that the Libby population is experiencing excess mortality from each of these asbestos-related

diseases. The ATSDR study found the occurrence of mesothelioma in Libby at about 1 death/per 2000 people versus an expected occurrence of approximately 1 case per million in the general US population. Several more cases of mesothelioma among current or former Libby residents have recently been submitted by treating physicians and strongly suggest that the actual mesothelioma risk is substantially higher than that reported by ATSDR. (Attachment 38) While it is clear that those in Libby are experiencing increased risk of asbestos-related malignancies, the specific radiographic abnormalities associated with their increased risk is presently unknown.

Grace comments that pleural plaques, one of the asbestos-related radiographic abnormalities observed among Libby residents, are not associated with lung cancer, citing Partanen 1992 (Attachment 39), Hillerdal 1997 (Administrative Record Supplement #484253), Churg 1998 (Administrative Record Supplement #487085), Edelman 1988 (Attachment 40), and Weiss 1993 (Attachment 41). Although, Grace misrepresents Hillerdal's (1997) (Administrative Record Supplement #484253) conclusions about asbestos exposure and increased risk of lung cancer which actually states that "there is an increasing body of evidence which indicates that asbestos at low exposure levels produces a slight increase in the relative risk of lung cancer even in the absence of asbestosis," the data is inconclusive with respect to the association between radiographic findings of circumscribed pleural disease and lung cancer, unlike that for asbestos exposure in general. The risk for lung cancer associated with parenchymal fibrosis is well documented in the medical literature. About 1% of the participants of the Libby medical screening program had asbestos-related parenchymal fibrosis reported by at least 2 B-readers.

Non-occupational asbestos exposures have clearly been shown to increase the risk for mesotheliomas. A recent case-control study found significantly increased risks from environmental asbestos exposure among residents living near an asbestos cement factory in Italy (Magnani 2001) (Attachment 42). A multicentric study performed by researchers in Spain, Italy, and Switzerland found that low-dose exposures to asbestos at home or in the environment carries a significant risk of mesothelioma (Magnani 2000) (Attachment 43). Significantly elevated mesothelioma rates were also found among residents living in Manville, Somerset County, New Jersey, the location of the largest asbestos manufacturing plant in North America (Berry 1996) (Administrative Record #338256). Another study found significantly increased rates of mesothelioma from environmental

asbestos exposure among residents living near a crocidolite mine in Australia (Hansen 1993) (Administrative Record #371372). In this study the authors found that the rate of mesotheliomas increased significantly with time from first exposure, duration of exposure, and cumulative exposure. Thus, cumulative exposures beginning in childhood are of great concern. A review of cases of mesothelioma occurring in individuals younger than 40 years old found that the median age of initial exposure was 10 years of age and a median duration of exposure was 120 months. The median latency between initial asbestos exposure and diagnosis of mesothelioma was 19 years (Kane 1990) (Attachment 44).

Grace also argues that pleural plaques and diffuse pleural fibrosis are not associated with mesothelioma citing comments from Andrew Churg (a Canadian researcher) in 1998. This assertion is contradicted by several studies performed in other countries which have found an increased risk of malignancy associated with circumscribed pleural disease. Hillerdal (1994 Pleural Plaques and Risk for Bronchial Carcinoma and mesothelioma; Chest 1994;105:144-50) (Attachment 45) reported in a 1994 study of 1588 Swedish men with 84% having only occasional low intensity asbestos exposure, found that pleural plaques alone on CXR indicate a significant exposure to asbestos, with a statistically significant increased risk for mesothelioma (1/1700 per year) and possibly also for bronchial carcinoma. The risk for mesothelioma observed by Hillerdal 1994 was smaller than that estimated by Edge (Edge JR. Asbestos-related disease in Barrow-Furness. Env. Res. 1976;11:244-7) (Attachment 46) in 1976 who found that plaque carriers had a risk of developing mesothelioma which was 1/377 per year. The difference may be due to the Edge cohort having been exposed to amphiboles and having potentially higher exposures. Of note, the expected occrrence of mesotheliomas in the United States is approximately 1 case per 1 million people per year.

Not only have pleural plaques been associated with an increased risk of mesotheliomas, but exposure to tremolite asbestos, in particular, has been associated with increased risk of mesotheliomas in a nonoccupationally exposed population (Luce D, Bugel I, Goldberg P, Goldberg M, Salomon et. al. Environmental exposure to tremolite and respiratory cancer in New Caledonia: a case-control study. Am J Epidemiol 2000 Feb 1;151(3):259-65) (Attachment 47).

It is interesting to note that Grace cites Dr. Churg, a Canadian researcher, to support thier argument that pleural disease is not associated with an increased risk of malignant mesothelioma. The Canadian researchers have focused on groups of workers and residents living around chrysotile mines in Quebec, Canada which do not appear to have the highly increased mesothelioma rates observed in other asbestos-exposed populations, especially those exposed to amphiboles. However, a number of studies by Dr. Churg and others have concluded that observed variation in risks of mesothelioma, and other asbestos-related diseases, among chrysotile miners in Quebec appear to be due to exposure to tremolite asbestos present in certain ore products or at certain mine sites (Churg A. Chrysotile, tremolite, and malignant mesothelioma in man.Chest. 1988;93:621-28., (Attachment 48) and Churg A, Wright JL, Vedal S. Fiber burden and pattern of asbestos-related disease in chrysotile miners and millers. Am Rev Res Dis. 1993; 148:25-31) (Attachment 49). In another study, increased rates of pleural calcifications and mesotheliomas among certain groups of Quebec chrysotile miners and millers were thought to be due to exposure to fibrous tremolite present only in specific mining areas (McDonald AD, Case BW, Churg A, et. al. Mesothelioma in Quebec Chrysotile miners and millers: epidemiology and aetiology. Am Occup Hyg. 1997;41:707-19) (Attachment 50). Studies of mesothelioma rates by Canadian researchers among Quebec chrysotile miners and millers and Libby vermiculite miners and millers (Stayner L, Dankovic DA, Lemon R. Occupational exposure to chrysotile asbestos and cancer risk: A review of the amphibole hypothesis. Am. J. Pub. Hlth. 1996;86:107-114) (Attachment 9) provide direct epidemiologic evidence indicating that Libby amphiboles are more potent than chrysotile in inducing mesothelioma. These studies found that the percentage of deaths due to mesothelioma among Libby vermiculite miners and millers was 2.4%, approximately six times higher than the percentage (0.4%) reported in a study of Quebec chyrsotile miners and millers. Thus, not only is pleural disease associated with malignant mesotheliomas, but tremolite asbestos exposure in particular appears to be more potent or toxic than other forms of asbestos in causing this deadly disease.

(page 18)

Grace contends that a comment by Pat Cohan, clinical coordinator for the Center for Asbestos-Related Disease, supports the position that the presence of plaques on chest x-rays is not indicative of asbestos-related disease. As indicated above, the evidence is overwhelming that pleural plaques are associated with increased risk of asbestos-related impairment and malignancy. The notices sent to participants of the Libby medical screening indicated the presence of any type of lung abnormality, not just those related to asbestos. However, ATSDR and EPA relied on data indicating the presence of only asbestos-related abnormalities as identified by at least two of three B-readers. Thus, Ms. Cohan simply intended to indicate that any findings of abnormalities needed to be further reviewed by experts to determine which abnormalities were asbestos-related.

(page 19)

Grace also argues that ATSDR's findings on loss of pulmonary function supports the assertion that pleural plaques are themselves harmless. ATSDR found that being a former Grace worker or having worked with vermiculite at a non-Grace job was associated with moderate to severe restrictive abnormalities, after adjusting for those restrictive abnormalities that would be caused by smoking, body fat, chest surgery or age. Consequently, these findings support the determination that pleural plaques are not harmless.

Finally, Grace suggests that the findings of ATSDR are not clinically significant for those who have had significant historical exposure to asbestos. As previously indicated, this allegation is contrary to the facts. ATSDR found that of those having lung abnormalities, at least 50% had no occupational history with Grace. These lung abnormalities are associated with disease, functional impairment and increased risk of mesothelioma. As indicated by the mortality study, many of those exposed to Libby amphibole asbestos do die from the exposure.

IV. *Air Sampling In Libby (page 20)*

Grace argues that EPA's air sampling in Libby does not support past or future response actions by the Agency. While taken in a variety of settings, all of EPA's air sampling data is relevant to the actions EPA has taken to date and may take in the future. For example, all of the

air sampling performed by EPA shows that where there is a disturbance of solid media containing Libby amphibole asbestos we find respirable fibers in the air in close proximity to that solid media, regardless of specific location.

A. Conditions under which the air concentrations were monitored

Grace contends that EPA air monitoring is inadequate to support the ongoing removal action. In support of their argument Grace indicates that the MRI report (1982) contains no analytical results of "airborne fiber concentration in the residential area of Libby." However, EPA notes that on page 15 of the MRI report Table 4 clearly indicates the existence of a monitor (Station #9) in the trailer court, an area of Libby that was residential during the MRI investigation and remains residential today. Historical fiber concentrations in this residential setting measured by phase contrast microscopy (PCM) were 0.03 fibers per cubic centimeter (f/cc) as indicated in Table 45 on page 68 of the MRI (1982) document. Fiber concentrations in this range present residential cancer risks in the range of 1 excess cancer per 100 individuals using standard EPA risk methodology, a level far exceeding that normally considered sufficient for emergency response action by EPA. This risk estimate does not account for the possibility of non-cancer disease related to asbestos fiber exposure, thus underestimating overall health risk.

Grace indicates that it is neither aware of nor is in possession of Mr. Eschenbach's discussion of historical fiber concentrations in Libby air. (Attachment 51) However, Grace's own records are filled with this and additional evidence of elevated mineral fiber in ambient Libby air. Grace records indicate their awareness of fiber concentrations as high as 11.3 f/cc near the bag house, a Grace facility, which EPA believes is the Export Plant that lies immediately adjacent to a residential area of downtown Libby. Monitors placed by Grace at the "New Penny's Store", the "St. Regis Office Area" and the "Hospital Area" indicate airborne fiber concentrations of 0.67, 1.5, and 1.1 f/cc respectively. (See W.R. Grace Dust Survey - 1/13/76) (Attachment 52) Again, these airborne concentrations are sufficient to pose extreme risk to the residential population of Libby and are likely to have contributed significantly to the observable increase in asbestos-related death and disease in Libby today. Additionally, Grace representatives would be advised to take note of letters from J.W. Wolter to E.S. Wood (7/27/1981) (Attachment 53) and McCaig to

Geiger (7/8/81) (Attachment 54) indicating that results of a Grace investigation at the school track recorded 0.14 f/cc and 0.22 f/cc for two test runners on the track.

These above referenced Grace documents indicate fiber concentrations in Libby exceeded safe levels. To the degree that the plant operated continually, we would expect airborne concentrations exceeded safe levels on a regular basis. This is relevant to EPA's concerns and removal activities as it confirms and supports contemporary measurements conducted by EPA that demonstrate the same phenomenon. That is, disturbance of soils, vermiculite products, and other substances contaminated with Libby amphibole can not only cause extremely elevated airborne fiber concentrations, but that even limited exposure to airborne fiber can result in significant morbidity and mortality. There is no logical reason to believe that the disease outcome of fiber exposures occurring today in Libby would differ from those which occurred 10, 15 or 20 years ago.

(page 21)

Grace argues that "there has been no elevation of ambient levels of airborne asbestos in Libby before, during, or after EPA's removal actions." Clearly the above-cited references demonstrate that this statement is simply untrue. However, for the purpose of present removal actions, EPA has not contended that ambient air concentrations in Libby exceed risk-based limits. Rather, EPA's investigations have clearly demonstrated that; 1) Grace failed to control waste products from the mine and processing plants, as well as ore concentrate, 2) that these materials have been widely distributed throughout the City of Libby and surrounding areas including on roadbeds, in residential gardens, on the school tracks, 3) disturbance of this material today results in elevated fiber exposure similar to exposures historically recorded by Grace and others, and 4) exposure to this material is clearly associated with radiologically-defined lung abnormalities and increased morbidity/mortality among the residential population.

At all properties where EPA has undertaken a response action there has been amphibole asbestos contamination at the surface under conditions where exposures may have occurred on a daily basis. Furthermore, at some locations EPA has chosen to remove subsurface source materials because it was reasonable to assume that exposures in the future would be likely. For

example, at the High School, surface contamination was found under the bleachers, around the track and in the locker room, concession stands and equipment room. Regular maintenance of the track, field and sprinkler system have occurred, and the exposure would have likely occurred regularly if cleanup had not been performed. Thus, the highly contaminated tailings under the pavement at the track was not "inaccessible."

Grace argues that no person has had a daily, residential exposure to emissions of amphibole asbestos fibers from Rainy Creek Road. Unfortunately, exposures occurring at the Parker residence prior to EPA action have proved this assertion incorrect. EPA has measured exposures resulting from traffic on Rainy Creek Road in the vicinity of the Parker's home and business. Since the Parkers lived and worked on this property, their exposure would be continuous on a daily basis except for vacations. It should also be noted that the Parker's children and grandchildren were exposed at this home by emissions from Rainy Creek Road. (Attachment 55)

Grace's concern that EPA's use of the personal air sample data taken from workers at EPA's removal actions overstates realistic exposures is unfounded. First, while the response actions may have been large in scope, these types of activities are likely to occur on a smaller scale in the future. While lesser quantities of soil may be moved, it is unlikely that the precautions EPA took to minimize the suspension of respirable fibers would occur. Therefore, the levels of amphibole asbestos in the air during such activities may actually be higher than those experienced by the response workers. For example, the Parkers and their employees planted trees on a daily basis in contaminated soils, swept amphibole laden dust covered floors, and installed sprinklers at the Screening Plant. None of these activities were performed with dust control measures or personal respiratory protection. In addition, the levels experienced by workers at the response action were similar to those identified by EPA during activities normally undertaken by residents such as digging, rototilling, sweeping or driving over contaminated media. In fact, it is EPA's observation that any contact activity with vermiculite based, amphibole contaminated media will generate elevated airborne asbestos fiber levels. As previously stated, these exposures can be associated to asbestos-related disease.

(page 22)

Grace argues that EPA's data do not provide support for the theory that attic insulation is the source of indoor air levels of asbestos. EPA's contention is simply that such insulation, just like any other Libby vermiculite media, creates high airborne levels of amphibole asbestos when disturbed by human activity.

Grace indicates that it believes that EPA's data does not support the decision to perform response activities at the Siefke property. EPA has reviewed Grace's concerns and finds them based on misinterpretations of the data and of the rationales for cleanup. EPA's sampling found asbestos fibers in dust, air and solid media in and around the Siefke home. As will be discussed below, the fiber size distribution found at this location is similar to that found elsewhere in Libby, both currently and in the 1970s. These fibers, whether long or short, are associated with lung disease. Thus, the fact that fibers may have been more than 5um or less than 5um, while pertinent to the risk presented by each fiber relative to other fibers, is not paramount to the cleanup decision. Both Ray and Trudy Siefke have asbestosis. While one cannot determine that this disease was caused by the presence of fibers in their own home (as opposed to other contaminated areas in Libby that they may have visited), their illnesses raise the level of concern about the presence of those fibers as both a potential cause of, and further exacerbating factor for the Siefkes' current condition. Therefore, the Agency's action was prudent.

Grace's assertion that the levels of amphibole asbestos found in settled dust within the Siefkes', and other Libby homes, do not warrant action is contradicted by Grace's own expert R.J. Lee. When asked to comment on the finding by private scientific testing firms that levels of asbestos were as high 79,000 f/cc inside apartments around the World Trade Center, Dr. Lee reportedly said "These dust levels are extraordinary. I think you'd have to recommend, based on these numbers, that they be professionally cleaned." (Attachment 56) EPA dust sampling at the Siefkes' and in several other Libby homes has found levels of amphibole asbestos in settled dust at this order of magnitude and much higher.

As to Grace assertion that there are misinterpretations of the data, EPA addresses the following five points. 1) Amphibole asbestos fibers identified by AHERA counting rules are, in fact, confirmed amphibole asbestos fibers. This method uses TEM with independent confirmation

of fiber type (via XRD or EDS) and thus indicate the presence of asbestos. Contrary to Grace's statements that only one air sample showed asbestos fibers, six samples from the Siefke home and three from the decon station where Siefke materials were being handled contained asbestos fibers. 2) Grace misuses the tabulation format used in presenting the data sets. In fact, asbestos fibers with a diameter greater than 0.5um are counted in almost all risk assessment methodologies, because they meet the PCME definition of >0.25 um width, >5um length, and an aspect ratio of >5:1. Therefore, these fibers do contribute to the risk as the site. In fact, three ISO analyses of air samples showed fibers greater than 5um, not one. 3)After the scenario testing was done on June 5th and 6th, EPA decontaminated the house on the 7th and ran clearance samples on the 8th. It appears that Grace is including in its counts analyses of samples taken during and after EPA's cleaning of the house on the 7th and the 8th. Thus, it would not be expected that these eleven samples would show asbestos fibers. 4) At the time EPA made its decision to take response actions at the Siefke property, the data showed amphibole asbestos fibers in three of five dust samples. 5) Grace raises concerns about the presence of chrysotile fibers found in some samples. EPA cannot understand Grace's point. None of the response action decisions were based on the presence of chrysotile asbestos.

(page 23)

Grace states that EPA's data does not show that the presence of vermiculite in exterior soils at the Brownlee residence results in interior dust/or air contamination. EPA documented the presence of a vermiculite pile at the Brownlee residence and cleaned up the pile. EPA took no response actions within the Brownlee home. Thus, Grace's comment is irrelevant.

Grace contends that elevated levels of asbestos will only be detected as a result of direct contact with vermiculite "associated with large-volume handling of previously inaccessible subsurface materials, or infrequently through activities that disturb surfaces with asbestos-containing dusts." While EPA agrees that direct contact with Libby vermiculite will result in the release of high levels of respirable amphibole asbestos fibers, EPA strongly disagrees that a "large-volume" of material is necessary to cause such a release. In fact, Grace's own studies from the mid 1970s through the mid 1980s (e.g., drop tests, friability determinations, simulated attic

tests, barrel transfer tests, etc.) show that the small amounts of the material release significant concentrations of amphibole asbestos fibers. Independent investigators have also identified and quantified the propensity for bulk materials to release fibers to the air. These investigators inadvertently encountered Zonolite insulation while conducting standard chrysotile asbestos abatement in a Canadian government facility. After careful (TEM) analysis of bulk Zonolite, asbestos concentrations in the solid phase were found to be on the order of 0.006 %. However, when disturbed, the material released copious quantities of asbestos fibers to the air. Airborne asbestos concentrations reached levels as high as 173 fibers/cc during normal activity. These levels are greater than 1700 times the federal occupational exposure limit. (Attachment 57) EPA's data also supports this fact. EPA also disagrees that inaccessible subsurface materials played a major role in EPA's determination. As previously stated, at every location that EPA took a response action there existed surface concentrations of vermiculite containing amphibole asbestos. Subsurface contamination was removed only where future exposure was likely.

B. Relative absence of the long, thin fibers thought to be of greatest significance toxicologically

(page 24)

Grace clearly misunderstands both Dr. Weis' risk calculations, and his discussion of the Berman-Crump risk model. To clarify, all decisions based on Dr. Weis' risk calculations in the July 9, 2001 memo relied on the EPA's standard asbestos risk model contained in the Integrated Risk Information System (IRIS). The appropriate size cut off for fibers included in the calculations done by this model are those longer than 5.0 um, not 10 um. In this model all asbestos fibers, regardless of morphology and mineralogy, that are longer than 5.0 um (with an aspect ratio >3:1; width >0.25um) are given an equal weight of toxicity. Hence, distinguishing fibers that are greater than 10um has no relevance to the IRIS analysis. Dr. Weis' discussion of Berman and Crump simply reflects that there are alternative risk assessment models proposed to evaluate asbestos risk. The approach taken by the Berman-Crump model is somewhat different than that in IRIS. This model proposes different risk-slope factors based on both mineralogy and morphology. This model proposes that in general amphibole asbestos is more potent than

serpentine asbestos, and that longer fibers are more potent than shorter. Thus, while all asbestos fibers > 5um are given some risk value, amphibole asbestos fibers >10um are given the greatest weight. If one were to use the Berman-Crump risk model almost any detection of an amphibole fiber >10um would yield a risk value greater than 1E-03. Any discussion of Berman-Crump model must acknowledge that where fibers greater than 10um exist, the risk-slope factor is nearly three orders of magnitude greater than that contemplated under IRIS where no such distinction is made.

EPA has been analyzing the size distribution of amphibole asbestos fibers in a variety of environmental settings which include both air and solid media. The results of this analysis are consistent throughout the environmental settings. The fiber size distribution of amphibole asbestos seen in samples collected from today are consistent with those reported by Grace in the 1970s. (See Yang, April 8, 1976, Characterization and Preparation of Respirable Sized Tremolite Fiber and Vermiculite For Animal Studies (Attachment 58) and William E. Smith, May 25, 1978, Final Report on Biologic Tests of Samples 22260p5 and 22263p2 (Attachment 59) and Weis memorandum entitled " Addendum Supporting and Clarifying the Libby Risk Memorandum of December 20, 2001 and Associated Libby Risk Memorandums. (Attachment 60) Since disease present today in Libby was likely caused by exposures during the 60s, 70s and 80s (due to the latency period), the fiber size distribution which caused that disease is also present in Libby today. (See 12/20/01 Weis Memo - Attachment 61, as well as Attachment 60) Thus, it is reasonable to construct two theoretical explanations for this occurrence of disease: Either the relatively small fraction of long fibers observed by EPA and Grace are in fact extremely potent, or the shorter fibers are in fact contributing to toxicity that has been so readily observed in Libby. Either way, the amphibole asbestos seen in Libby today is the same material that has lead to death and disease among so many in the past and continues today.

Lastly, one should recognize that there have actually been quite a few long fibers found in conjunction with activities in Libby. Grace argues that only 21 fibers longer than 10um were seen in the 143 samples reported from the mine. Grace fails to note that most of the samples collected were stationary ambient air samples taken when no activity was taking place. If one were to look at the data from samples collected when site activities were taking place one would see that the

levels of asbestos always go up. The Agency has consistently made the observation that the activity or behavior taking place controls the significance of the exposure as much, if not more than the total concentration of asbestos present. (See also Attachment 57) Indeed, in EPA's scenario testing, we typically find that 15% of the fibers are greater than 10 um (with over 50% greater than 5 um, consistent with Grace work in the 1970s and NIOSH work in the 1980s). As to the observation that the EPA has only seen fibers >10um in approximately 3% of the residential settings sampled, the Agency would point out that we have only conducted clean-ups in roughly 3% of the properties sampled.

C. Improper analytical procedures and use of counting data

(page 24)

Grace contends that EPA has inappropriately calculated PCMEs if those findings are going to be compared to the OSHA PEL. EPA counted fibers in a manner that would allow it to determine risk pursuant to the Agency's peer reviewed IRIS model. The PCME finding determined by using TEM represents an actual count of asbestos fibers that people were exposed to. Despite the fact that this count may vary from that found by PCM, which would likely miscount asbestos fibers in this situation, a qualitative comparison to the PEL is useful as a relative guage of the magnitude of the exposure. As indicated in Dr. Weis' memorandum, the identified exposures exceeded the PEL. Even if occupational exposure limits were complied with, risk would be well above EPA's risk guideline. Dr. Weis also discusses in a chart on page 6 of his memo actual PCM results at the Export/Screening plants which exceed the OSHA PEL. Thus, no matter which procedure is used, the fiber levels are unsafe and required EPA intervention.

As the facts show, PCM is not a reliable tool for determining actual airborne asbestos exposure. ISO 10312 TEM, which was developed by the International Standards Organization, adopted for use by the European Union, is peer reviewed and has, after consultation with a variety of experts in the field, been determined to be the an appropriate tool for measuring asbestos concentrations.

The use of TEM and the associated ISO 10312 structure enumeration approach has advantages as it allows for possible future consideration of the effect of structure length and width

on toxicological potency rather than structure dimensions as artificially defined by the limitations of phase contrast microscopy (PCM). The rationale for the complete measurement and identification of fibers by TEM is based on recent epidemiologic data (including those studies published since the adoption of the IRIS file). Furthermore, use of the ISO 10312 counting procedure is based on the assessment of the structures using TEM which incorporates fiber characteristics of structures too thin to be detected using PCM (which contribute significantly to potency). Nonetheless, EPA is ensuring that data will be collected and analyzed in Libby in such a manner so that the protocol structure approach as well as the IRIS-based (PCME) cancer risk estimates can be appropriately applied.

(page 26)

Grace's circular arguments concerning the appropriateness of using TEM or PCM are dizzying. On the one hand, Grace criticizes the Agency for not using PCM measurements in making risk evaluations, but mere paragraphs later quotes the IRIS model about the unreliability of PCM. "Measurements by PCM which are made in conditions where other types of fibers may be present may not be reliable." While Grace may choose to stand motionless in its criticism of each available technique, EPA has actually moved forward in evaluating both techniques and using the information available in an informed decision-making process. EPA has found that no matter how Libby asbestos is measured, the concentrations found at cleanup locations have exceeded commonly acceptable health based limits (i.e., 10E-6 cancer risk), sometimes by several orders of magnitude.

D. Inappropriate statistical analysis of the data

(page 27)

Grace contends that Dr. Weis uses inappropriate statistical analysis of the data in his memorandum. In particular, Grace takes issue with Dr. Weis' use of maximum PCM values in his risk assessment analysis. Dr. Weis calculates both a maximum likelihood exposure (MLE) based on the arithmetic mean and a reasonable maximum exposure (RME) (based on either the 95% upper confidence limit (UCL) or maximum value measured). It has been common practice in

EPA risk assessment to employ both the MLE and RME to estimate risks to human receptors. Incorporation of both estimates in Dr. Weis' memorandum is neither inappropriate nor unusual. RME exposures are commonly developed by EPA calculating the 95% UCL on the arithmetic mean exposure point concentration. In cases where data is below the reporting limit for the analytical method, EPA guidance requires employment of one half (½) the analytical reporting limit as a surrogate estimate. RMEs calculated by this method that do not exceed the maximum value of the data set are employed as exposure point concentrations. If the estimate by this method exceeds the maximum value recorded for the set, then the risk assessment policy recommends defaulting to the maximum value of the data set for the RME. This methodology is described in the very document which Grace cites (Exhibits 28 and 29) and is available for further clarification.

(page 28)

Grace also expresses consternation about Dr. Weis' treatment of non-detect data. The guidance document cited by Grace and attached as Grace Exhibit 29 (Risk Assessment Guidance for Superfund Volume I, Human Health Evaluation Manual, Part A, December 1989) discusses four treatments of non-detect data based upon the nature and type of calculation being made. Non-detects may be inputed as ½ the sample quantification limit, as the sample quantification limit, as zero, or excluded from the data set for purposes of the risk calculation. Dr. Weis used each of these treatments in accordance with the guidance in making his calculations. EPA suggests that Grace re-review the guidance to understand that the different methods are used in particular situations which are reflected in the different calculations performed by Dr. Weis. It should be noted that where zero is substituted for a non-detect analytical value, the risk calculation tends to underestimate exposures since the true value may be anywhere between zero and the reporting limit. Thus, estimated exposures and risks presented in Dr.Weis' memorandum are likely to be low and actual exposures and risks may be higher than those recorded.

E. The Supplemental Administrative Record Does Not Contain Key Information to Allow Meaningful Comment

(page 28)

Grace argues that the administrative record supplement is incomplete because it does not specify which data points were used for the calculations performed by Dr. Weis and does not contain backup laboratory information for the data. All of the data on which Dr. Weis relied was included in the supplement to the administrative record. Dr. Weis' memorandum describes the rationale and methodology for his calculations, as does the guidance referenced above. The procedures for the collection and analysis of samples in Libby, including QA/QC requirements, were discussed at length in the Phase 1 and Phase II sampling plans, which were included in the administrative record.

While the NCP allows for a broad array of documents to be included in the administrative record, it does not suggest that every piece of backup documentation be included as well. To do as Grace suggests would subject the public to a record which would be incomprehensible to the lay person, too large to navigate and would subvert the intended purpose of informing the public in a meaningful manner. EPA has business practices that assure that data is accurate. One purpose of such business practices is to ensure consistent handling of data and obviate the need for the inclusion in the administrative record of detailed backup for each piece of information. (Grace has now had access to all of the information relating to EPA's sampling data and response actions, even that not in EPA's possession.) Another purpose of such business practices is to ensure that the On-Scene Coordinator can delegate some of these QA/QC functions to appropriate staff and focus his attention on major issues, thus ensuring an expedited response action in time critical situations. The On-Scene Coordinator relies on consolidated layers of information produced by experienced individuals, including information about the validity of data.

(page 29)

Grace suggests that because EPA identified "isolated nuggets of what appear to be "tremolite rocks"" as part of its evaluation of the need for action at the high school, there is the appearance that EPA is making decisions when "preliminary data are not even available." EPA

has been sampling and analyzing solid media in Libby since November of 1999. EPA, in consultation with USGS and on-site professional geologists, has sampled and analyzed tremolite rocks at Libby in sufficient quantity to understand their general characteristics and likely range of asbestos concentrations. EPA has sufficient data to understand the implications of the presence of tremolite rock without analyzing each and every rock. EPA's identification of these rocks is assisted by the professional geologist. Grace has obtained the USGS data in question.

F. Response to Mr. Cohn's July 26, 2001 Letter Regarding Grace's Comments About the Original Administrative Record

(page 30)

Grace submits rebuttal comments to EPA's responses to Grace's comments on the administrative record for the export and screening plants. While the NCP provides no opportunity for a party to make such comments, EPA replies below, incorporating by reference its July 2001 response to Grace.

Item J, page 8

(page 30)

While the use of infrared technology is relatively new to the asbestos assessment field, Grace underestimates its use. At this time, EPA is aware that infrared is used by USGS, Environmental Monitoring Support Laboratory, Reservoir Laboratory and some universities.

Item K, page 9

(page 30)

While Grace implies that it was using OSHA ID 160, a peer reviewed and published OSHA methodology, it is interesting to note that this methodology was first established in its peer reviewed form in 1988. EPA's initial comment concerning Grace's use of a non-peer reviewed "discriminatory technique" concerned Grace testing occurring in the 1970s. Thus, there was no peer-reviewed OSHA ID 160 when Grace was performing the testing. In addition, the OSHA

methodology requires that fibers be counted as asbestos unless the analyst is absolutely sure that the fiber can be counted as something else.

Item M, page 10

(page 30)

Grace continues to criticize EPA's TEM analysis, asserting that the filters used by EPA did not "contain uniformly distributed fibers." The method used by EPA (i.e., ISO 10312) does not rely on uniform distribution of fibers on the filters, but is premised on an asymmetrical distribution of those fibers. This asymmetrical distribution is inherent in the design of the filter cowls used for collection of all asbestos samples and is accounted for in the analysis of the sample. Thus, filter segments would not be expected to have identical fiber concentrations as Grace contends and EPA's findings are appropriate. Grace also implies that EPA's analyses had false positives. EPA's QA/QC process requires that our labs analyze blanks on a periodic basis to identify false positives. No false positives were identified.

P. 14

(page 31)

Grace continues to suggest that weather conditions in Libby would diminish exposure and that human activity has no bearing on exposure. The data obtained by EPA during active sampling shows there is re-entrainment of asbestos fibers in solid media into the breathing space of those involved in activities which disturb the asbestos-contaminated solid media. This finding is consistent with observations by independent investigators and even Grace scientists. Thus, when weather is dry and people undertake activities that disturb vermiculite, raw ore, or ore concentrate, there is an observable increase in airborne fiber concentrations.

G. A. Inadequate Study Design

1. Scenario 1

(page 31)

Grace contends that EPA's scenario 1 testing was invalid because: 1) there were not enough homes sampled; 2) participation was voluntary; and 3) the amount of air to be sampled was impractical. EPA agrees that the larger the sample size, the greater the degree of confidence one can have in the results. However, there are limits based on budget, exigent circumstances and practicality. EPA has only so much money it can spend on Libby and needs to ensure that an appropriate percentage is used for actual cleanup work. EPA's work in Libby is a time-critical response to an immediate threat. Thus, EPA must limit the time spent in sampling and analysis to that absolutely necessary so that the contamination can be addressed as quickly as possible. Practically, there are only so many homes with the appropriate attributes and willing volunteers. The study objectives were clearly defined prior to initiating the study, the samples were carefully collected and the analyses performed were sufficient to understand the effect of the activity studied in scenario 1. In implementing scenario 1 testing, EPA selected a group of homes which met the attributes necessary for testing. Volunteers were then sought from the owners of these homes. Thus, the voluntary nature of the participation of each household created little bias in the results. Finally, the amount of air to be collected was planned to meet the study objectives. No problems were encountered during the air sampling and an appropriate volume of air was collected and analyzed.

2. Scenario 2

(page 32)

Grace argues that the study design for the scenario 2 sampling was not conducted in a uniform manner across subjects and sites and therefore did not reflect "typical cleaning practice". EPA used cleaning practices which were determined, in part through homeowner discussions, to be typical of the homes which were tested. While Grace indicates that "dry sweeping is not an effective way to clean dusty floors" these homeowners, like the rest of the American public, do

use a broom in their home. EPA's intention with respect to the Phase 2 scenario 2 sampling was to determine a plausible upper-bound exposure RME for residents in a few situations that were likely to occur in Libby. These measurements were made with great care to assure highest quality sampling and analysis according to the Phase 2 plan. Such exposure measurements are completely consistent with the Agency's Risk Assessment Guidance for Superfund. (See http://www.epa.gov/superfund/programs/risk/ragsa/index.htm.) Unequivocally, the scenario 2 data indicates that individuals in residential environments in Libby can be exposed to asbestos at levels which create greater than acceptable cancer risk.

3. Scenario 3

(page 32)

Grace contends that EPA's Scenario 3 testing was too limited, particularly as it relates to homes which would be considered to have "background" concentrations. As indicated in the previous response, the phase 2 investigation was intended to provide information about plausible residential exposures in Libby. As clearly outlined in the Sampling Plan for the project, it was not the objective of Phase 2 to conduct a City-wide survey of ongoing residential exposure measurements. Rather, the Phase 2 investigation was conducted to determine whether residents may currently be exposed to asbestos in their homes and to quantify the magnitude of those exposures if they were encountered. The results indicate that exposures may indeed occur at levels greatly exceeding commonly acceptable levels. The findings of the Phase 2 Libby investigation are further supported by similar yet independent data collected by EPA's Office of Pollution Prevention and Toxic Substances (OPPTS) and EPA Region 10.

V. *Screening Risk Levels Using Libby Area Air Sampling (page 33)*

EPA is not required by the NCP to perform formal risk assessments to support the need for time-critical removal actions. Section 415(b)(2) of the NCP establishes the criteria which EPA must use to determine whether a removal action is appropriate. As discussed in the action memorandum (July 2001) and in other parts of the administrative record, EPA has appropriately

made that determination. Nonetheless, EPA has used the risk assessment process to further evaluate the need for response.

Grace argues that "screening-level risk estimates should not be used to support expensive removal actions". In the case of the screening-level risk estimates for Libby, commonly accepted cancer risk levels (10E-4 to 10E-6) were exceeded, in some cases, by several orders of magnitude. Non-cancer risks were not even quantified in the subject risk memorandum even though there is an apparent epidemic of asbestos-related lung disease in Libby. Risk estimates are, by nature, uncertain. However, the excessive risks estimated from the Phase 2 data, coupled with a plethora of medical information demonstrating severe human toxicity resulting from exposure to Libby amphibole, warranted immediate action.

Grace also argues that exposure assumptions were inconsistent and "worst case". Dr. Weis used exposure assumptions derived from national guidance documentation (Exposure Factors Handbook) and interviews with Libby residents. During the Phase 2 investigation, EPA recorded exposure concentrations exceeding occupational exposure limits for both short and long term safety of healthy adult workers. Exposure of infants, the elderly, or any residents not protected by occupational safety regulations to these excessive concentrations of asbestos is unacceptable.

A. Rainy Creek Road

1. Location of Asbestos Samples - Impact on Rainy Creek Road Exposure Scenario

(page 33)

Grace contends that the exposure scenarios along Rainy Creek Road were inappropriate. EPA strongly disagrees that theoretical exposure scenarios estimated along Rainy Creek Road are unreasonable. Until EPA intervened, residents who live directly at the base of Rainy Creek Road were heavily exposed to asbestos-contaminated ore, ore concentrate and contaminated soils. EPA is aware of serious actual exposures experienced by these residents, including their children and grandchildren (see Attachment 55). Moreover, areas along the road could very well be developed in the future, resulting in uncontrolled exposures to airborne asbestos.

To monitor possible residential exposure along Rainy Creek Road, EPA placed monitoring devices on adjacent properties. Actual data collected from these monitors demonstrated regular exceedances of airborne exposure limits when Grace failed to implement required dust suppression measures. Data provided to Grace demonstrates exceedences for the dates of October 30, November 10, 11, 13, and 14 (see correspondence of 11/16/00). Availability of such environmental data is usually preferable to use of the PUFF model or other theoretical exposure assessment tools. Note that EPA periodically recorded exceedances of airborne asbestos concentrations along Rainy Creek road during one of the wettest seasons of the year.

2. *Representativeness of Air Sampling Data for Long-Term Exposure Assessment*

(page 34)

Grace argues that the exposure duration used along Rainy Creek Road is too long and that actual exposures would have been of shorter duration (2 hours/day vs. 24 hours/day). Grace ignores the fact that there are indeed residents who live and work full-time near Rainy Creek Road. The Parkers were on their property most of each day, either working at their nursery or living in their home. In addition to the ongoing exposure from the road itself, data from the phase 2 investigation clearly indicate that, once a residence is contaminated by external or internal sources of asbestos, indoor exposures may exceed those received outside. Such re-entrained asbestos fiber may remain a source of ongoing exposure indefinitely. Thus, the exposure estimates for dust from Rainy Creek Road were reasonable and possibly underestimated possible exposures indoors. Data collected at a residence at the base of rainy creek road as well as other residential areas of Libby supports this conclusion.

Importantly, the risks presented in the screening level risk assessment do not include an assessment of noncancerous disease due to asbestos exposure along Rainy Creek Road. Given that there is significant evidence that Libby amphibole causes asbestosis as well as cancer, the subject risk memorandum is likely to have underestimated rather than overestimated risk.

3. *Residential Exposure Assumptions for Hypothetical Residents in Rainy Creek Road*

(page 35)

Grace argues that the duration of exposure used to assess risk along Rainy Creek Road was inappropriately long. In fact, demographic information for the Libby vicinity coupled with resident interviews indicates that the Libby population is stable and, in some cases families have remained in the City for several generations. Furthermore, extended family visitations may result in longer than average exposure durations in Libby and cumulative exposure in different sections of town. This was certainly the case for the residents at the base of Rainy Creek Road, where 3 generations of family members regularly shared the home. Thus, it is plausible and reasonably protective of public health for estimates of exposure duration to be extended in the Libby vicinity.

4. *Revised Lifetime Cancer Risk Estimates*

(page 36)

Grace estimates of risk presented in the context of their comments to EPA are neither consistent with Agency guidance for upper bound screening level risk assessments nor protective of public health. Nonetheless, even Grace risk estimates demonstrate exceedance of the 10E-6 risk threshold for cancer (risk estimates within the 10E-4 to 10E-6 range require EPA application of risk management decisions). EPA notes that Grace risk estimates fail to even mention non-cancer risks associated with exposure to Libby amphibole despite clear evidence that non-cancer mortality is 40 to 60 times higher in Lincoln County than elsewhere in the Country.

B. *Residential Scenarios*

(page 38)

Grace's concern that EPA was measuring peak concentrations during residential scenario testing and would thus bias its risk results is misplaced. EPA did not rely on peak measurements, in fact designing the study to avoid temporal peaks. The analysis was based on the longest full-period sample for each activity. A reference to a maximum was the maximum average value

obtained for a specific activity scenario as between the three homes tested, not between fluctuations within the sampling period.

Kinetics of airborne fiber concentrations following mechanical disturbance of asbestos-contaminated materials has been investigated by Grace. Details of such measurements are available in the administrative record.

VI. Alternative Analytical Techniques (page 39)

Grace argues that EPA cannot use bulk samples to provide support for the decision to pursue removal actions. Data available in the peer reviewed open literature and clearly referenced in Dr. Weis' memo (Addison, 1988), demonstrate that fibers in solid media are easily released to the air when disturbed. Addison demonstrated that soils contaminated with asbestos at only a fraction of a percent may release fibers to the air at concentrations well above acceptable limits. (Administrative Record Supplement # 485923) In addition to the work conducted by Addison, Grace conducted extensive measurements quantifying the release of asbestos fiber from solid media. EPA's phase 2 investigation and the Grace findings confirm the Addison work. During the phase 2 investigation, EPA conducted detailed human exposure measurements in Libby residences to verify these findings under conditions commonly encountered by residents. Therefore, EPA disagrees that there is no method for estimation of risks from exposure to asbestos-contaminated solid media. While measurement relating asbestos-contaminated solid media to human exposure may be indirect, these measurements are scientifically sound and clearly indicate high levels of human exposure under conditions presently encountered by residents in Libby.

VII. Processing Plants
(page 39)

Grace contends that EPA's reference to the Jorgensen case is inappropriate, as his deposition testimony indicates that he was exposed to multiple sources of asbestos at the Western Minerals facility. In addition, Grace challenges the finding that Mr. Jorgensen had asbestosis.

The Jorgensen case involves an individual who died at age 42 with asbestosis and asbestos-related lung cancer stemming from childhood exposure to Libby vermiculite piles. According to the selective portions of Mr. Jorgensen's deposition submitted by Grace, Mr. Jorgensen played in piles of stoner rock (containing high levels of amphibole asbestos) and played on and around bags and blocks potentially containing chrysotile asbestos located in the plant and in boxcars. While Mr. Jorgensen recounts that the bags and blocks were covered in dust, there is no information to indicate whether the dust came from operation of the plant, from unloading of boxcars or hopper cars filled with vermiculite, or some other source. However, Mr. Jorgensen's pathology report is indicative of the dust to which Mr. Jorgensen was exposed. Mr. Jorgensen's autopsy report (Fargo Clinic 6/22/91, Attachment 62) clearly documents findings of bilateral pleural plaques on his right and left diaphragms. A followup pathology report (Duke University Medical Center 9/24/91, Attachment 63) concluded that Mr. Jorgensen had parietal pleural plaques and histological findings consistent with the diagnosis of asbestosis. Lung specimens taken during his autopsy showed actinolite and tremolite fibers in his lungs with a markedly increased tremolite content. Of the 20 fibers evaluated by EDXA, 18 were tremolite and 2 were actinolite. Lung content was within the range found in patients with asbestosis with up to 3810 asbestos bodies/gram wet lung (normal range 0-20) and up to 142,000 uncoated fibers/ gram wet lung. In addition to asbestosis, Dr. Roggli, a leading expert in the field of asbestos pathology, thought that Mr Jorgensen's adenocarcinoma was related, at least in part, to his prior history of exposure to asbestos living in the vicinity of an asbestos plant and playing on piles of vermiculite contaminated with tremolite fibers as a child.

While much lower in lung asbestos burden, the types and percentages of asbestos fibers in Mr Jorgensen's lungs are consistent with pathology results of a former Libby vermiculite worker, Mr. McNair (WRG Meeting 17 October 1983, Patrick Sebastien and Ben Armstrong, Attachment 64), and a 65 year-old accountant who worked for 2 months at age 17 in a vermiculite expansion plant. The results of Mr. McNair's lung evaluation showed 49.7 million uncoated fibers /gram dry weight with 87% of the fibers being tremolite, 3.5% other amphiboles, and no significant amount of chrysotile present. It was commented that the uncoated fiber size distribution in Mr. McNair's lung was remarkably similar to air samples taken from Libby.

VIII. *International Conferences (page 40)*

On pages 17 and 40 Grace makes reference to comments made at a few conferences on asbestos exposure. While EPA does not have the resources to check what each presenter's oral comments where, EPA does understand the positions reflected by these individual's peer reviewed publications. Some of the representations made by Grace are not reflected by the published material. For instance, Grace indicates that Hillerdal believes that "plaques are in themselves harmless". In fact, in a 1994 peer reviewed publication, Hillerdal states that "pleural plaques on chest roentgenogram indicate significant exposure to asbestos, with an increased risk for mesothelioma and possibly also for bronchial carcinoma." Earlier comments in this response provide more information on the published materials of these presenters.

IX. *Dr. Whitehouse (page 42)*

A discussion of Dr. Whitehouse's findings is presented earlier in these comments. A preliminary report of his findings is attached to this response.

X. *Academic Papers (page 43)*

Grace clearly has its own interpretation of the published academic papers. It is not surprising that Grace's view varies from that of EPA and many of the scientists in the field.

XI. *Barbanti Litigation (page 43)*

Grace has provided no information regarding the importance of the remainder of the Barbanti litigation. Thus, EPA does not understand the relevance of Grace's inclusion of the documents in its comments, and therefore in the Supplemental Administrative Record. Nonetheless, EPA has reviewed some of the materials relating to Drs. Lee, Corn and Hughson. EPA is unconvinced by their testimony. In fact, it appears that some of the assertions made by one or more of these experts is unsupported. (It is EPA's understanding that there has not been a ruling in that case at this time.)

Summary

EPA has fully reviewed Grace's extensive comments. None of the information provided indicates that the decisions EPA has made have been arbitrary and capricious. Likewise, those decisions are not inconsistent with the NCP.